John C Anderson
United States Attorney

September 13, 2019

Re: A[redacted] G[redacted] Wa[redacted]

Mr. Anderson:

At your request I reviewed the available medical treatment records and autopsy report regarding A[redacted] G[redacted] Wa[redacted]. In addition, I reviewed the transcripts of interviews of Farrol and Jamil Louis-Jacques. You have requested my expert medical opinion regarding several questions which I will outline in some detail below.

By way of background, I am board certified in pediatrics (1990), as well as in Adult Neurology with Special Competence in Child Neurology (1995, recertified 2007 and 2018). Currently I am Professor with tenure of Neurology and Pediatrics at the University of New Mexico Health Science Center. I have over 2 decades of experience as a child neurologist, and have been Director of the Division of Pediatric Rehabilitation (1998-2003) and of the Division of Pediatric Neurology (2010-2019) at UNM. As part of my daily clinical work I have followed many children with neonatal encephalopathy, epilepsy and cerebral palsy. This report is being generated during my annual leave time and opinions expressed in this report are my own and do not represent the University of New Mexico or any other agency.

Let me summarize pertinent medical aspects of [redacted] history as I understand them. [redacted] was born to a 43 year old mother at 42 weeks gestation. Pregnancy was reportedly uncomplicated and a home delivery was planned. After 23 hours of home labor without delivery, an ambulance was called and [redacted] was born in the vehicle en route to the local public hospital (Grady). He was intubated on arrival to the Emergency Room due to poor respiratory effort and copious meconium was noted. Birthweight was normal at 3.175 kg. [redacted] was extubated and then reintubated again for bradycardia and oxygen desaturations then admitted to the NBICU where he underwent the standard 3 day cooling protocol. On the second day of life an EEG captured electrographic seizures and phenobarbital was started. At one week of age a brain MRI showed findings consistent with "severe HIE." It was noted that he was hypotonic and had poor feeding, and after 3 weeks he was discharged home on NG feeds and the

anticonvulsant phenobarbital.

One week later (now 1 month old) he was readmitted with respiratory stridor and during this PICU admission bronchoscopy and laryngoscopy disclosed laryngomalacia, tracheomalacia, an elliptical cricoid with mild narrowing with evidence for gastroesophageal reflux. Note was made that had been tried on oral feeds at home. After one week in the hospital he was discharged home on reflux management and mother already had a breast pump to allow breast milk to be given via NG.

At 7 weeks he was seen in the outpatient ENT clinic by Dr Prickett because of a failed newborn hearing screen and sensorineural hearing loss (mild right, mild-moderate left) was diagnosed. Hearing aids were recommended, as well as consults by ophthalmology and genetics. An outpatient echocardiogram was done about this time showing a PDA with some left to right atrial shunting but no pulmonary hypertension. This compared to an initial echocardiogram at 1 week showing mild pulmonary hypertension.

As indicated in later chart notes, by 3 months Early Intervention therapies had begun; an SLP outpatient encounter at 3 months documents a primary goal of feeding without aspiration. Also at 3 months [redacted] was provided hearing aids. Just before 4 months he was doing well and SLP recommended gradually transitioning to full oral feeds. At 4 months [redacted] was seen again by SLP and a GI physician whose notes indicate that [redacted] was immediately started on all oral feeds and had actually been doing well, therefore the recommendation was to continue oral feeds with reflux precautions. At this time a Barium Swallow test was done by radiology/SLP showing normal swallow function without reflux. [redacted] was also seen by Dr Simon (a pulmonologist) at this time who states he is starting to make some developmental progress, and refers to his lack of vaccines which was apparently because mother was worried about his frequent illnesses and not for religious reasons.

At 5-6 months of age there are copies of clinic notes from SLP (working on safe swallowing) and audiology (hearing aid checkup) and ENT.

An ER visit was prompted by a seizure with fever at 6 months of age. Notes indicate that the primary care physician recommended weaning off phenobarbital several months previously, and [redacted] had 2 spells that day, each several seconds of leg rhythmic jerking, felt to be a seizure. Exam noted otitis media and he was discharged home on rectal diazepam and antibiotics.

Some time after the seizure, he was restarted on phenobarbital and a concern was raised regarding premature suture closure prompting a head CT scan at 11 months of age; this showed "global cerebral atrophy, likely caused by early in life hypoxic ischemic event."

At almost 1 year of age on 7-30-15 [redacted] was admitted via the emergency room for status epilepticus of over 45 minutes duration requiring several doses of lorazepam then phenobarbital loading to stop. At that point he was seen for the first time by the neurology service as an inpatient consult; note was made of reportedly good medical compliance on 2 mL BID (16 mg/d ~ 1.7 mg/kg/d) which is a low dose with the serum phenobarbital level correspondingly low at < 5. BMI is 17 (low) and legs were noted to have increased tone and DTRs. An overnight EEG showed diffuse slowing but no seizures, phenobarbital was increased to a more appropriate dose of 3.5 mL BID (28 mg/d ~ 3 mg/kg/d) and after several days on the ventilator he was successfully extubated and discharged home. He was apparently scheduled to see neurology several months later but there is indication this appointment missed.

1 month later at almost 13 months of age he was seen by neurosurgery because of concern re: small fontanelle and premature suture closure; it was felt this was due to poor brain growth not synostosis, that his skull was not dysmorphic and surgical intervention was not recommended. The measured OFC was 43.5, just under the 2nd percentile.

Over the following year [redacted] continued to have periodic visits with SLP and ENT and then at approximately 23 months of age on 6-16-16 there was a formal assessment by Dr Adams-Chapman and the developmental team that included the Bayley assessment by a PhD developmental psychologist (Dr Carter). It was noted that [redacted] was receiving PT, OT and SLP weekly. The OFC was 44.3, falling further away from the 2nd percentile. BMI was 13.7 which is lower than the prior year. Abnormal tone and brisk reflexes were noted, consistent with cerebral palsy. Bayley showed social skills at 12-18 months, expressive language 11 months, receptive language 10 months, fine motor 5 months, gross motor scattered skills below 6 months.

One month later, at 24 months of age, Abdul was seen in the child neurology clinic; there may be an earlier visit although this was not documented in the records that I have access to. Examination by the neurology team at that point showed spastic diplegia with a left cortical thumb, hyperreflexia with crossed

adductors and upgoing plantar responses, all c/w their diagnosis of spastic CP. Note was made that mother stopped phenobarbital several months prior after talking to PCP; for this reason an EEG was obtained the following month read as normal, and in a f/u neurology clinic one month afterwards it was recommended that [redacted] stay off AEDs unless further seizures occur.

Unfortunately at 31 months of age in March 2017 seizures again occurred; [redacted] had seized an hour prior to presentation in the ER where they stopped only after multiple meds (midazolam, lorazepam, fosphenytoin). He was seen by neurology, another EEG showed abnormal lack of well-defined background activity but no ongoing seizures, and after an overnight hospitalization he was discharged home on levetiracetam.

Several days later [redacted] had a routine follow up appointment in the Developmental Clinic. It is stated that the mother was following through on all recommendations and caring for [redacted] well. Exam has OFC at 45.7 cm which would represent a continued gradual fall-off lower than the 2nd percentile. The examination included an Ireton Developmental Chart which indicated good social and motor developmental progress (now sitting, feeding himself finger foods, smiling, waving bye…) with less language improvement. Thus, a referral for alternative and augmentative communication was made. Spasticity was recognized as a growing problem which might benefit from Botox, and it was recommended he f/u in the CP clinic for this, in addition to seeing orthopedics and have an evaluation for equipment (knee immobilizers, seating).

Finally, the last note available for review in the medical record was from Dr Weissman on May 1st 2017, a child neurologist with expertise in cerebral palsy. [redacted] was approximately 32-33 months of age. Dr Weissman indicates that [redacted] has been seizure free, was receiving weekly developmental therapy (PT, OT, SLP), had appropriate braces, tolerated 1 hour daily in a stander, and had an examination showing spastic diplegia with what may be a right hemiparesis. Recommendations included increasing levetiracetam slightly to a more appropriate dose and Botox was scheduled to the legs, with a 3 month follow up planned.

The 2 interview transcripts were also reviewed. I believe these include reference to [redacted] coming to the New Mexico household in the winter. In clarifying this after speaking with Mr George Kraehe by phone, I understand that [redacted] was abducted from his home and taken to The Taos area on approximately December 1st, where he lived for several weeks before dying in some sort of

prayer ritual likely at the end of December 2017. Indications from the interview transcripts are that [redacted] may not have been getting his prescription medication, not receiving adequate nutrition (one interviewee recalls food shortages and being hungry), and that there were prayer rituals 5/day each of perhaps one hour duration during which time [redacted] was held down despite crying and resisting, sometimes leading to a possible seizure with eyes rolling back and foam coming out of his mouth. The actual death is not well described; the interviewee describes a prayer ritual that he heard but didn't see where (presumably) [redacted] crying stopped and someone said his heart had stopped.

Questions I was asked to comment on are the following:

1. A[redacted] G[redacted] medical diagnosis and treatment history.

[redacted] history suggests no prenatal problems, consistent with his normal birth weight, however perinatal complications resulted in a precipitous ambulance delivery with immediate respiratory distress and bradycardia with copious meconium on intubation. He fulfilled criteria for neonatal cooling and underwent the now standard 3 day cooling protocol, showing electrographic seizures on the 2nd day of life. MRI at a week of age showed findings consistent with "severe HIE." This presentation and clinical course is consistent with what is now termed neonatal encephalopathy (American College of Obstetrics and Gynecologists, Task Force Report (2014)Vol 123;4:896-901). Also consistent with this clinical course was a head CT done at 11 months showing global atrophy. The head circumference at 1 month was at the 25th percentile and gradually fell off the chart to under the 2nd percentile by 2 years of age. Thus, it appears there was a single devastating event around the time of birth causing brain damage and resulting in subsequent lack of normal expected brain growth, becoming more evident with age.

Consequences of [redacted] brain injury include epilepsy, developmental delay, dysphagia, gastroesophageal reflux disease, and poor weight gain. As a result of these medical problems, [redacted] was heavily involved in the medical system. Weekly early intervention services (including PT, OT, SLP) began at 3 months and continued through at least until he was almost 3 years old. He was followed by multiple specialists such as ENT, GI, pulmonary, audiology, orthopedics, neurology and developmental specialists in addition to his primary care physician. He had several EEGs, an MRI and CT of his brain, formal developmental

assessment, swallow and hearing studies, and several ER visits with PICU admissions for prolonged seizures.

Thus, [redacted] medical diagnosis was neonatal encephalopathy resulting by 32 weeks of age in cerebral palsy, epilepsy, poor weight gain and developmental delay. His treatment history was comprehensive and appropriate involving a large number of medical and rehabilitation therapists. Indeed, in the last Developmental Clinic note at ~ 32 months of age, it appears with this medical support and attention by his mother, [redacted] had made nice progress in several developmental domains, now sitting, feeding himself finger foods, smiling, and waving goodbye. One can reasonably assume that with continued appropriate care, [redacted] life expectancy would have been well into adulthood (Brooks, Recent Trends in Cerebral Palsy Survival, DMCN (2014)56:1065-1071).

2. The medical treatment A[redacted] G[redacted] required prior to his death.

As outlined in treatment history, above, [redacted] received a number of necessary and in some cases life-saving medical treatments. He initially was fed via NG tube because of aspiration risk (which frequently leads to pneumonias). [redacted] had sensorineural hearing loss with hearing aids necessary to maximize opportunity for language development. Seizures became a life-threatening problem (July 2015, March 2017) which didn't stop until multiple medications were given on 2 separate occasions in the emergency room leading to requiring ventilatory support in the PICU. As motor difficulties and tone abnormalities became evident, [redacted] fit the diagnosis of spastic cerebral palsy and required Botox injections and adaptive equipment (stander, gait trainer, braces). Furthermore, [redacted] development was severely delayed and required formal developmental assessments with ongoing frequent therapy services (PT, OT, SLP) and nutritional recommendations for his poor weight gain. It was likely gratifying to the team working with [redacted] and his mother that, over time, he was able to demonstrate nice developmental gains, particularly in the domains of motor and social function.

3. The reasonable probable outcome to A[redacted] G[redacted] if he did not receive required medical treatment.

Assuming there was no medical treatment provided after December 1, 2017, over a subacute period of time measured in weeks to a

month, one could reasonably expect several sequelae. This includes issues such as: 1) [redacted] spasticity could get worse, resulting in painful muscle cramping spasms. 2) Nutritional support likely would be poor, causing further weakening of [redacted] muscles. He may not have been fed with appropriate precautions as indicated in one of the interviewees stating that he ate what everyone else was eating – for example, a note from March 2017 while still getting medical attention in Georgia indicates that he was getting feeding therapy and was still unable to take full foods such that he could not chew pasta for example. 3) Therefore, he would be at risk for aspiration and respiratory distress as a result, compounding his weakness. 4) Perhaps the most dangerous problem facing [redacted] if he was denied medical care would be seizures. The half-life of levetiracetam is ~ 6-8 hours, thus it would be effectively "out of his system" and subtherapeutic within several days (Patsolos, Clin Pharmacokinet 2004;43(11):707-24) if this were stopped. Such an abrupt withdrawl of anticonvulsant medication places him at risk of seizures, which in his case in the past did not stop until multiple medications were given in an emergency room setting.

Death can occur in patients with epilepsy through several mechanisms. Sudden Unexpected Death in Epilepsy (SUDEP) is often a post-ictal phenomenon occurring during sleep (Devinsky, Lancet Neurology 2016;15:1075-1088). More relevant to [redacted] case, however, is death as a direct result of a prolonged seizure (status epilepticus). In a hospital setting, status epilepticus is associated with ~ 1% death rate, often associated with cerebral palsy, pneumonia and respiratory failure (Loddenkemper, PLoS One 2012;7:10). Out of the hospital, without appropriate medical care, death related to status epilepticus is certainly higher. Ongoing seizure activity is exhausting, causing weakness and increasing difficulties breathing. Compounding this weakness would be airway obstruction, such as would be the case if secretions or vomitus were in the oropharynx. Death from ongoing seizure activity can occur as a primary cardiac event or respiratory arrest.

Thus, without medical care, [redacted] would likely have experienced increasing spasticity and pain, increasing weakness related to poor nutrition and possibly aspiration, and eventually, status epilepticus.

4. The medical treatment A[redacted] G[redacted] received prior to December 1, 2017.

All medical care was appropriate and indicated. Indeed,

clinicians also commented on the good job mother was doing caring for [redacted].

5. The medical treatment A[redacted] G[redacted] received after December 1, 2017.

I have no records indicating that [redacted] received medical care after December 1, 2017.

6. A[redacted] G[redacted] cause of death.

While it is of course impossible to state with certainty, the transcripts indicate that he likely died during one of the many prayer rituals he was subjected to. Several factors would have contributed to his death as follows.

[redacted] had spastic cerebral palsy. Spastic muscles are weak, and as with most children of his age with cerebral palsy, [redacted] was underweight. Thus he had generalized weakness making it difficult to fully protect his airway. Furthermore, indications are that [redacted] was held down in the supine position, crying during these rituals. This is a position that leaves the airway poorly protected and any secretions or vomitus (there is indication in the transcripts that "foam came out of his mouth") would interfere with breathing. Crying indicates energy expenditure thus with time muscle weakness would increase, further interfering with effective respiration while held down during these rituals.

What was described in the transcripts would be consistent with a generalized seizure occurring during the prayer ritual, with eyes rolling back and unresponsiveness. This is not unexpected given the circumstances. It seems likely [redacted] anticonvulsant medication was not given, and after several days his risk for seizures would increase. Furthermore, seizures also occur more often with stress including psychological stress, such as might occur while being held down despite crying in protest. Because [redacted] had a history of seizures that didn't stop (until given multiple medications in an emergency room setting) it is likely that at some point, when a seizure occurred, it did not stop on its own. With ongoing seizure activity, in a weakened condition and unable to protect his airway, it is more likely than not that a respiratory arrest would ensue leading to cardiac arrhythmia, asystole and death.

Also possible is that death occurred without a seizure occurring. As noted, [redacted] was a frail young man with spastic cerebral palsy, likely in poor nutritional status who would have difficulty protecting his airway. If forced down during the prayer rituals in the supine position, crying against his will, eventually he would tire and airway obstruction likely would occur, particularly if as indicated some type of liquid (secretions, vomitus?) were in his mouth. Choking and breathing obstruction would quickly cause further weakness leading to respiratory arrest, becoming quiet, and as time passed without breathing there would be a cardiac arrest and death.

John Phillips, MD